09/844508
A9 #17

WEST Search History

DATE: Wednesday, June 04, 2003

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L3	l1 same L2	165	L3
L2	remodel$	11128	L2
L1	chromatin	3429	L1

END OF SEARCH HISTORY

WEST

Print Selection

Help | Clear | Cancel | Print | Print First Page

Select?	Document ID	Section(s)	Page(s)	# Pages to print	Database
☑	20030041343	all	all	28	USPT,PGPB,JPAB,EPAB,DWPI
☑	20020064523	all	all	42	USPT,PGPB,JPAB,EPAB,DWPI
☑	20020106789	all	all	* 118	USPT,PGPB,JPAB,EPAB,DWPI
☑	6268173	all	all	* 85	USPT,PGPB,JPAB,EPAB,DWPI
☑	6350572	all	all	30	USPT,PGPB,JPAB,EPAB,DWPI
☑	6159684	all	all	12	USPT,PGPB,JPAB,EPAB,DWPI
☑	6444421	all	all	41	USPT,PGPB,JPAB,EPAB,DWPI
☑	6183965	all	all	38	USPT,PGPB,JPAB,EPAB,DWPI
☑	6214588	all	all	* 120	USPT,PGPB,JPAB,EPAB,DWPI
☑	6248520	all	all	* 71	USPT,PGPB,JPAB,EPAB,DWPI
☑	5972608	all	all	19	USPT,PGPB,JPAB,EPAB,DWPI
☑	US5972608A	all	all	N/A	USPT,PGPB,JPAB,EPAB,DWPI

Note: Print requests for more than **49** pages are denoted by '*' and are in red.

Building	Room	Printer
cm1	11e14	gbldptr

Main Menu | Logout

09/844508
AA # 17

=> s snp or (single nucleotide polymorphism#)
L1 25548 SNP OR (SINGLE NUCLEOTIDE POLYMORPHISM#)

=> s remodel?

L2 89626 REMODEL?

=> s l1 and l2

L3 92 L1 AND L2

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4 34 DUP REM L3 (58 DUPLICATES REMOVED)

=> d l4 ibib abs 1-34

L4 ANSWER 1 OF 34 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:203290 HCAPLUS
DOCUMENT NUMBER: 138:232951
TITLE: Targeted modification of chromatin structure with fusion proteins containing DNA binding domain and chromatin ***remodeling*** complex protein
INVENTOR(S): Wolffe, Alan P.; Wolffe, Elizabeth J.; Collingwood, Trevor; Snowden, Andrew
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 56 pp., Cont.-in-part of U.S. Ser. No. 844,508.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 6
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003049649	A1	20030313	US 2002-84826	20020224
US 2002115215	A1	20020822	US 2001-844508	20010427

PRIORITY APPLN. INFO.: US 2000-200590P P 20000428
US 2000-228523P P 20000828
US 2001-844508 A2 20010427

AB A fusion protein and compn. for targeted modification of chromatin structure within a region of interest in cellular chromatin are provided. The fusion protein comprises a DNA binding domain and a protein component of a chromatin ***remodeling*** complex. The compn. comprises the fusion protein and addnl. proteins such as transcription factors and histone (de)acetylase. Use of such fusion proteins and compns. for facilitating processes such as, for example, transcription and recombination, that require access of exogenous mols. to chromosomal DNA sequences, is disclosed. Thus, the method was demonstrated by regulation of human VEGF gene expression in HEK 293 cells by a fusion protein consisting of a modified SP1 DNA-binding domain fused to human BAF155 subunit of the BRM/BRG chromatin ***remodeling*** complex. Similar studies with zinc finger DNA binding domains fused to human gene MDB1 (methyl-binding) protein, to ISWI chromatin ***remodeling*** ATPase, to steroid receptor coactivator 1 (a histone acetyltransferase), and to a DNA N-methyltransferase and regulation of the human erythropoietin gene are described.

L4 ANSWER 2 OF 34 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2003139955 EMBASE
TITLE: Pharmacogenomics of congestive heart failure.
AUTHOR: Baliga R.R.; Narula J.
CORPORATE SOURCE: R.R. Baliga, Division of Cardiology, Department of Medicine, University of Michigan, Ann Arbor, MI 48103, United States. rrbaliga@umich.edu
SOURCE: Medical Clinics of North America, (2003) 87/2 (569-578).
Refs: 33
ISSN: 0025-7125 CODEN: MCNAA
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
022 Human Genetics
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The future of pharmacogenomic therapy for CHF will require the determination of key genes that are critical in mediating cardiac ***remodeling*** [28]. Cardiac ***remodeling*** includes several processes including cardiac myocyte hypertrophy and apoptosis. Determination of genes that turn on and off the processes of hypertrophy and apoptosis may allow the future clinician to determine which patients with dilated cardiomyopathy would require antiapoptotic therapy, therapy directed to reducing cardiac myocyte hypertrophy, therapy directed to influencing cell survival, or a combination of such therapies. One strategy for identifying culprit heart failure genes is to compare expression profiles and cellular responses in human and mouse models of disease (Figs. 1 and 2). A high-throughput screen using common transcripts in the mouse and human models of heart failure is the initial step. This is followed by evaluation of the function of these novel genes by using the genes in mouse culture with the gain of function assay or the loss of function assay (antisense nucleotides). Other steps in the confirmation of the culprit gene include the use of transgenic animals to verify their individual contribution to the pathogenesis of CHF [29]. One group of investigators [30] using some of these techniques, particularly large-scale automated DNA sequencing, generated over 40,000 expressed sequence tags (ESTs) from human heart cDNA libraries. In addition, they retrieved over 40,000 ESTs from public databases. They found that, from the total of 84,904 ESTs, about 55% matched known genes, or approximately one third, matched other ESTs, and approximately 12% did not match any known sequences. Many of the latter are novel genes, and further efforts have to be made to verify their contribution to the pathogenesis of heart failure. Using high-density oligonucleotide arrays, one group of investigators found that several genes were altered in the failing heart, and they grouped these into five clusters. The clusters included (1) genes that encode contractile and cytoskeletal proteins (ie, SLIM1, .beta.-acting, and myomesin MLC2); (2) genes encoding proteins that influence the disassembly and degradation of myocardial proteins (ie, ubiquitin, gelsolin, and .alpha.1-antichymotrypsin); (3) genes encoding proteins of the mitochondria (ie, aldose reductase, ATP synthase .alpha.-subunit, and TIM17 preprotein translocase); (4) genes encoding stress proteins (ie, .alpha..beta.-crystallin and .mu.-crystallin); and (5) genes encoding proteins that influence protein synthesis including EF-2 and transcription factor homologue-HBZ17. Although the precise role of these proteins in CHF remains to be characterized, these genes should enhance our understanding of signaling pathways in the cardiac myocyte and, consequently, promote the development of newer therapies for CHF including gene delivery depending on pharmacogenomic profile (see the article by Dr. Hajjar et al elsewhere in this issue). Fast forward to the year 2010. A typical patient with CHF will require a blood test to determine his or her gene profile for the management of the disease. A deletion/insertion of the polymorphism in the ACE gene indicating that the patient is salt-sensitive suggests that salt restriction and diuretic therapy are more important in such a patient than in one who is not salt-sensitive [31-33]. A bradykinin-receptor polymorphism suggesting that the patient may develop a cough with ACE-inhibitor therapy means the clinician will have to avoid an ACE inhibitor. If the patient has beta-receptor polymorphisms suggesting that he or she will respond to beta-blocker therapy, then the patient may have only beta-blocker and diuretic therapy rather than the whole complement of ACE-I, digoxin, and diuretics. Depending on the genetic variant of CYP2D6, the clinician can decide whether the patient requires only .beta.-1 blockade with metoprolol XL or requires .alpha.1/.beta. blockade with carvedilol. In a different scenario, if the patient has pulmonary hypertension and a receptor polymorphism of the endothelin receptor, then the clinician may decide on an endothelin-receptor antagonist as first-line therapy (although, at this point of time, endothelin receptor antagonists have not been shown to be beneficial when added to standard therapy). Therefore, tailored therapy will not only allow a choice of which drug is more effective, but it will

AUTHOR: Vadas P.; Browning J.; Edelson J.; Pruzanski W.
CORPORATE SOURCE: Division of Immunology, Department of Medicine, Wellesley
Hospital, 160 Wellesley St. East,Toronto, Ont. M4Y 1J3, United States
SOURCE: Journal of Lipid Mediators, (1993) 8/1 (1-30).
ISSN: 0921-8319 CODEN: JLMEEG
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 002 Physiology
005 General Pathology and Pathological Anatomy
022 Human Genetics
029 Clinical Biochemistry
052 Toxicology
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Human non-pancreatic PLA2 has been the object of intense scrutiny for a
relatively short period of time. Its role in physiology remains enigmatic. While PLA 2 may serve to ***remodel*** or remove peroxidised or senescent phospholipids, the enormous magnitude of its upregulation during
infectious or inflammatory episodes is consistent with a role in host defense. However, the nature of this role remains elusive. Attempts to relegate this enzyme to the genre of acute phase reactants have not been helpful in unravelling its role. Difficulty in obtaining adequate amounts of native ***snp*** -PLA2 prior to the availability of recombinant ***snp*** -PLA2 led to the widespread use of snake venom homologs, particularly in studies of the biology of PLA2. This review has underscored the pitfalls inherent in that approach given the major differences between some venom PLA2s as compared to ***snp*** -PLA2. In
addition, it bears reiterating that the complex composition of venom allows for potentiation of PLA2 activity by other constituents present in venom. Whether human host defense networks employ this interactive strategy is largely unknown. Nonetheless, in spite of these reservations, some very compelling data have emerged in recent years implicating ***snp*** -PLA2 in the initiation or potentiation of local and systemic inflammatory processes. These include sepsis and associated acute lung injury as well as inflammatory arthritides, with rheumatoid arthritis as the prototype. The mechanisms of ***snp*** -PLA2 homeostasis are considerably better understood, and it has become apparent that ***snp*** -PLA2 is an integral part of a larger network of proinflammatory cytokines, growth factors and lipid mediators. The interrelationship between the functions of secretory and cytosolic PLA2s remains to be defined. A number of selective PLA2 inhibitors have been identified which will allow for discrimination between the actions of these classes of PLA2. The availability of synthetic inhibitors in conjunction with endogenous modulators of PLA2s will shift the biology of
PLA2 from the realm of the inferential to that of the mechanistic.

=> d his

(FILE 'HOME' ENTERED AT 17:46:59 ON 12 JUN 2003)

FILE 'BIOSIS, EMBASE, MEDLINE, WPIDS, HCAPLUS' ENTERED AT 17:47:08 ON 12
JUN 2003
L1 25548 S SNP OR (SINGLE NUCLEOTIDE POLYMORPHISM#)
L2 89626 S REMODEL?
L3 92 S L1 AND L2
L4 34 DUP REM L3 (58 DUPLICATES REMOVED)

=> s transcription factor#
L5 274451 TRANSCRIPTION FACTOR#

=> s l1 and l5

L6 506 L1 AND L5

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7 295 DUP REM L6 (211 DUPLICATES REMOVED)

=> s l7 and py<2000

1 FILES SEARCHED...
3 FILES SEARCHED...
4 FILES SEARCHED...
L8 35 L7 AND PY<2000

=> d l8 ibib abs 1-35

L8 ANSWER 1 OF 35 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:374073 BIOSIS
DOCUMENT NUMBER: PREV200200374073
TITLE: Functional genomics and DNA array techniques in atherosclerosis research.
AUTHOR(S): Hiltunen, Mikko O.; Niemi, Mari; Yla-Herttuala, Seppo (1)
CORPORATE SOURCE: (1) Department of Molecular Medicine, A.I. Virtanen
Institute, University of Kuopio, FIN-70211, P.O. Box 1627, Kuopio: Seppo.Ylaherttuala@uku.fi Finland
SOURCE: Current Opinion in Lipidology, (***December, 1999***)
Vol. 10, No. 6, pp. 515-519. http://www.co-lipidology.com/. print.
ISSN: 0957-9672.
DOCUMENT TYPE: General Review
LANGUAGE: English

L8 ANSWER 2 OF 35 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:2234 BIOSIS
DOCUMENT NUMBER: PREV200000002234
TITLE: Characterisation of the human snail (SNAI1) gene and exclusion as a major disease gene in craniosynostosis.
AUTHOR(S): Twigg, Stephen R.F.; Wilkie, Andrew O.M. (1)
CORPORATE SOURCE: (1) Institute of Molecular Medicine, John Radcliffe
Hospital, Headington, Oxford, OX3 9DS UK
SOURCE: Human Genetics, (***Oct., 1999***) Vol. 105, No. 4, pp.
320-326.
ISSN: 0340-6717.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The Snail family of proteins in vertebrates comprises two zinc-finger ***transcription*** ***factors*** , Snail and Slug, which are thought to be involved in the formation of the mesoderm and neural crest. Here, we
describe the isolation and characterisation of the human Snail (SNAI1) gene and a related Snail-like pseudogene, SNAI1P. SNAI1 spans approximately 6.4 kb, contains three exons and has a CpG island upstream of the coding sequence. A single transcript of 1.9 kb was detected in several human foetal tissues, with the highest expression in the kidney. The SNAI1 open reading frame encodes a protein of 264 amino acids containing four zinc-finger motifs that show 87.1% identity to mouse Snail (mSna). SNAI1 was mapped to chromosome band 20q13.1 and is likely to lie
between markers D20S109 and D20S196. Investigation of SNAI1 coding sequences by single-strand conformation polymorphism analysis excluded SNAI1 as a major disease gene in craniosynostosis. Two ***single*** ***nucleotide*** ***polymorphisms*** encoding synonymous amino acids
were identified in exon 2. The SNAI1P pseudogene was isolated, sequenced
and mapped to chromosome band 2q34.

L8 ANSWER 3 OF 35 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:320729 BIOSIS
DOCUMENT NUMBER: PREV199900320729
TITLE: Variation at the von Willebrand Factor (vWF) gene locus is associated with plasma vWF:Ag levels: Identification of three novel ***single*** ***nucleotide*** ***polymorphisms*** in the vWF gene promoter.
AUTHOR(S): Keightley, Angela M.; Lam, Y. Miu; Brady, Jolene N.; Cameron, Cherie L.; Lillicrap, David (1)

09/844508
Art #15

=> s chromatin
L1 121062 CHROMATIN

=> s remodelling

L2 17865 REMODELLING

=> s fusion

L3 488148 FUSION

=> s zinc finger

L4 24076 ZINC FINGER

=> s dna binding domain

L5 22882 DNA BINDING DOMAIN

=> s dbd

L6 3960 DBD

=> s l1 and l2

L7 957 L1 AND L2

=> s l1 and l2 and l3
L8 49 L1 AND L2 AND L3

=> s l1 and l2 and l4
L9 15 L1 AND L2 AND L4

=> s l1 and l2 and l5
L10 18 L1 AND L2 AND L5

=> s l1 and l2 and l6
L11 3 L1 AND L2 AND L6

=> s l8 or l9 or l10 or l11
L12 75 L8 OR L9 OR L10 OR L11

=> dup rem l12
PROCESSING COMPLETED FOR L12
L13 39 DUP REM L12 (36 DUPLICATES REMOVED)

=> d l13 ibib abs 1-39

L13 ANSWER 1 OF 39 MEDLINE
ACCESSION NUMBER: 2002396100 MEDLINE
DOCUMENT NUMBER: 22140045 PubMed ID: 12145209
TITLE: Targeting of SWI/SNF ***chromatin*** ***remodelling*** complexes to estrogen-responsive genes.
AUTHOR: Belandia Borja; Orford Rob L; Hurst Helen C; Parker Malcolm G
CORPORATE SOURCE: Institute of Reproductive and Developmental Biology, Imperial College Faculty of Medicine, Du Cane Road, London W12 0NN, UK.
SOURCE: EMBO JOURNAL, (2002 Aug 1) 21 (15) 4094-103. Journal code: 8208664. ISSN: 0261-4189.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200209
ENTRY DATE: Entered STN: 20020730
Last Updated on STN: 20020917
Entered Medline: 20020916
AB SWI/SNF complexes are ATP-dependent ***chromatin*** ***remodelling*** enzymes that have been implicated in the regulation of gene expression in yeast and higher eukaryotes. BRG1, a catalytic subunit in the mammalian SWI/SNF complex, is required for transcriptional activation by the estrogen receptor, but the mechanisms by which the complex is recruited to estrogen target genes are unknown. Here, we have identified an interaction between the estrogen receptor and BAF57, a subunit present only in mammalian SWI/SNF complexes, which is stimulated by estrogen and requires both a functional hormone-binding domain and the DNA-binding region of the receptor. We also found an additional interaction between the p160 family of coactivators and BAF57 and demonstrate that the ability of p160 coactivators to potentiate transcription by the estrogen receptor is dependent on BAF57 in transfected cells. Moreover, ***chromatin*** immunoprecipitation assays demonstrated that BAF57 is recruited to the estrogen-responsive promoter, pS2, in a ligand-dependent manner. These results suggest that one of the mechanisms for recruiting SWI/SNF complexes to estrogen target genes is by means of BAF57.

L13 ANSWER 2 OF 39 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:260995 BIOSIS
DOCUMENT NUMBER: PREV200200260995
TITLE: The genome-wide localization of Rsc9, a component of the RSC ***chromatin*** -remodeling complex, changes in response to stress.
AUTHOR(S): Damelin, Marc; Simon, Itamar; Moy, Terence I.; Wilson, Boris; Komili, Suzanne; Tempst, Paul; Roth, Frederick P.; Young, Richard A.; Cairns, Bradley R.; Silver, Pamela A. (1)
CORPORATE SOURCE: (1) Department of Biological Chemistry and Molecular Pharmacology, Harvard Medical School, Boston, MA, 02115: pamela_silver@dfci.harvard.edu USA
SOURCE: Molecular Cell, (March, 2002) Vol. 9, No. 3, pp. 563-573. http://www.molecule.org/. print. ISSN: 1097-2765.
DOCUMENT TYPE: Article
LANGUAGE: English
AB The cellular response to environmental changes includes widespread modifications in gene expression. Here we report the identification and characterization of Rsc9, a member of the RSC ***chromatin*** -remodeling complex in yeast. The genome-wide localization of Rsc9 indicated a relationship between genes targeted by Rsc9 and genes regulated by stress; treatment with hydrogen peroxide or rapamycin, which inhibits TOR signaling, resulted in genome-wide changes in Rsc9 occupancy. We further show that Rsc9 is involved in both repression and activation of mRNAs regulated by TOR as well as the synthesis of rRNA. Our results illustrate the response of a ***chromatin*** -remodeling factor to signaling cascades and suggest that changes in the activity of ***chromatin*** -remodeling factors are reflected in changes in their localization in the genome.

L13 ANSWER 3 OF 39 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:427488 BIOSIS
DOCUMENT NUMBER: PREV200200427488
TITLE: Functional analysis of sperm from c-mos-/- mice.
AUTHOR(S): Gross, Vera S.; Cooper, Geoffrey M. (1)
CORPORATE SOURCE: (1) Department of Biology, Boston University, 5 Cummington Street, Boston, MA, 02215: gmcooper@bu.edu USA
SOURCE: Molecular Reproduction and Development, (August, 2002) Vol. 62, No. 4, pp. 519-524. http://www.interscience.wiley.com/j pages/1040-452X/. print. ISSN: 1040-452X.
DOCUMENT TYPE: Article
LANGUAGE: English
AB The c-mos protooncogene, which is expressed predominantly in male and female germ cells, is crucial for normal oocyte meiosis and female fertility in mice. Inactivation of c-mos results in abnormal oocyte development and leads to ovarian cysts and tumors in vivo. In contrast to the severe effects of c-mos ablation in females, targeted inactivation of c-mos has not been reported to affect spermatogenesis in male mice. However, previously reported studies of male c-mos-/- mice have been limited to histological analyses of testes and in vivo matings, both of which are relatively insensitive indicators of sperm production and